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blood and urine. Unfortunately, body fluid levels of cotinine in nonsmokers have never been correlated precisely with ambient nicotine exposures, as Repace and Lowrey assume, and body fluid levels in nonsmokers are affected by ingestion of nicotine from the ordinary diet.

- The level of "obvious risk" generated for cotinine by the Repace-Lowrey model could be achieved through the ingestion of common foods alone, in the absence of ETS exposure.

- Cotinine is a biologically inactive and non-carcinogenic substance metabolized from many common foods. Is the Repace-Lowrey estimate of "excess risk" based on this substance meaningful?